TABLE 2. *Methods used in retrospective and cross sectional studies of peptic ulcer and smoking (cont.)*

| Author, year, country, reference | Sex | Number | Method of selection | Controls | | Comments |
|---|---|---|---|---|---|---|
| | | | | Number | Method of selection | |
| Kasanen and Forsström, 1966, Finland (*13*). | M | 43 Gastric. 57 Duodenal. | Successive male admissions with peptic ulcer treated at medical clinic or outpatient department of University Hospital. Only patients under 65 years of age or those who had been working were included. | 100 | Successive men treated at medical clinic who had no gastrointestinal symptoms or signs of CHD. | A special questionnaire was used for the interview. |
| Gillies and Skyring, 1968, 1968, Australia (*9*). | M and F | 100 Gastric. 50 Duodenal. | Patients with peptic ulcer were selected from hospital admissions in 1967. | 150 | Matched by age and sex from the same ward at the same time and with absence of signs or symptoms or past history of upper gastrointestinal disease. | Diagnosis well established with X-ray, gastroscopy, or surgery. |
| Gillies and Skyring, 1969, Australia (*10*). | M and F | 10 Gastric. 48 Duodenal. 18 Uncertain location. | 1,405 workers from a broadcasting company, a manufacturing company, and a bus company were interviewed for a history of peptic ulcer. | 100<br>1,329 | Two control groups:<br>1. 100 peptic ulcer patients previously reported by authors.<br>2. 1,329 workers without ulcer. | All information obtained by question card. All ulcers were proved by X-ray or surgery. |
| Monson, 1970, U.S.A. (*15*). | M and F | 52 Gastric. 452 Duodenal. 139 Not specified. | 643 physicians from Massachusetts who responded affirmatively to a questionnaire sent to them in 1967 asking how many had had a peptic ucer. | 625 | Controls were physicians without ulcer disease who were matched to ulcer patients by year of birth. | Diagnosis established by X-ray or surgery except for 46 "clinical" cases. |

TABLE 3.—*Summary of results of retrospective and cross sectional studies of peptic ulcer and smoking*

| Author, year, country, reference | Percent nonsmoker | | Amount of tobacco used | |
|---|---|---|---|---|
| | Cases | Controls | Cases | Controls |
| Barnett, 1927, U.S.A. (*2*). | Total ..... 18.0<br>Gastric ..... 15.0<br>Duodenal ..... 20.0 | 25.0 | | |
| Trowell, 1934, England (*21*). | Duodenal ..... 8.0 | 17.0 | Average number:<br>Cigarettes .. 12.0 per day<br>Pipe ..... 1.6 ounces per week | .....11.1 per day<br>.. 2.15 ounces per week |
| Allibone and Flint, 1958, England (*1*). | 38.0 | 54.0 | | |
| Doll et al., 1958, England (*7*). | Gastric:<br>Males ..... 1.3<br>Females ... 51.1<br>Duodenal:<br>Males ..... 2.1<br>Females ... 53.7 | 4.7<br>66.8<br><br>5.8<br>62.0 | Gastric: *Percent smoking >25 cigarettes per day*<br>Males ..... 10.6<br>Females .... 1.1<br>Duodenal:<br>Males ..... 10.2<br>Females .... 1.9 | 11.3<br>1.1<br><br>12.7<br>1.9 |
| Edwards et al., 1959, England (*8*). | *Percent of peptic ulcer by smoking category*<br>Never smoked ..... 6.0<br>Formerly smoked ..... 6.7<br>Cigarettes:<br>1–9 per day ..... 9.4<br>10–19 per day ..... 9.8<br>>20 per day ..... 12.0<br>Pipe ..... 6.5<br>Pipe and cigarettes ..... 8.5 | | | |

TABLE 3.—*Summary of results of retrospective and cross sectional studies of peptic ulcer and smoking (cont.)*

| Author, year, country, reference | Percent nonsmoker | | Amount of tobacco used | | |
|---|---|---|---|---|---|
| | Cases | Controls | | Cases | Controls |
| Kasanen and Forsstr5m, 1966, Finland (*13*). | "Peptic" ..... 10.0 | 40.0 | Cigarettes per day: | | |
| | | | <10 ..... 10.0 | | 7.0 |
| | | | 10–20 ..... 19.0 | | 17.0 |
| | | | 20 ..... 42.0 | | 26.0 |
| | | | >20 ..... 19.0 | | 10.0 |
| Gillies and Skyring, 1968, Australia (*9*). | Gastric ..... 18.0 | 44.0 | Mean number cigarettes per day: | | |
| | Duodenal ..... 62.0 | 71.0 | Gastric .... 23.3 | | 17.1 |
| | | | Duodenal .. 23.2 | | 23.0 |
| | | | Duration of smoking (years): | | |
| | | | Gastric .... 30.2 | | 28.0 |
| | | | Duodenal .. 24.2 | | 28.2 |
| Gillies and Skyring, 1969, Australia (*10*). | Gastric ..... 17.9 | 55.6 | | | |
| | Duodenal ..... 36.6 | | | | |
| Monson, 1970, U.S.A. (*15*). | Duodenal ..... 32.1 | 46.7 | Percent smoking >20 cigarettes per day | | |
| | Gastric ..... 19.2 | | Age: | *Gastric* | *Duodenal* |
| | Not Specified .. 43.2 | | 20 ..... 38.8 | 27.3 | 30.1 |
| | | | 30 ..... 45.7 | 43.0 | 47.1 |
| | | | 45 ..... 60.2 | 49.5 | 46.9 |
| | | | 60 ..... 54.1 | 40.4 | 44.0 |

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# CHAPTER 7

## Tobacco Amblyopia

## Contents

## Tobacco Amblyopia

Tobacco amblyopia (tobacco-alcohol amblyopia) is that syndrome of visual failure occurring in association with the use of tobacco, with or without the concurrent use of alcohol, and with or without concurrent nutritional deficits. The disease has a subacute onset, leading to a loss of visual acuity and color perception (*12*). It is characterized by centrocecal scotomas which are bilateral but not necessarily symmetrical and which have sloping diffuse edges and by the presence of nuclei of denser visual loss within the large scotomas (*22, 23*). Such visual impairment is not unique to tobacco amblyopia, as it is also seen in neurodegenerative disorders, such as Leber's hereditary optic atrophy (*7, 25*).

Clinical information on tobacco amblyopia has appeared in numerous articles throughout the past century. This information has been reviewed by Silvette, et al. (*17*) and, more recently, by Dunphy (*5*). Pure tobacco amblyopia (TA), that is amblyopia unassociated with excessive alcohol intake or the exposure to other toxins, is rarely seen in the United States today (*12*). Walsh, et al. (*23*) have observed that when TA is found it is usually present in association with nutritional or idiopathic vitamin deficiencies. Victor (*22*) recently observed that the type of visual defect seen in tobacco amblyopia may be found in clinical circumstances in which tobacco is clearly not a causative factor. He questions whether TA is distinguishable from other forms of amblyopia.

The prevalence of this disorder has been variously estimated in the past at from 0.5 to 1.5 percent of all eye clinic patients (*20, 23*). However, currently in the United States, it appears to be a rare condition. Silvette, et al. (*17*) have observed that the incidence of tobacco amblyopia appears to have decreased substantially during the past decades. Other authors (*3, 15*) have also commented on this trend. Although reference has been made to the increased frequency of certain types of tobacco usage in patients with this disorder, adequate population studies with proper controls have yet to be performed. The association of this disorder with the use of tobacco is strengthened by the frequent clinical observations of improvement following the cessation of smoking although improvement has been noted by some to occur without cessation.

Research into the pathogenesis of tobacco amblyopia has cen-

tered upon the interrelationships of cyanide metabolism, vitamin $B_{12}$, and other vitamin deficiencies. Three reviews of this material have recently appeared (*1, 12, 22*). Numerous studies reviewed in these articles suggest that tobacco amblyopia may result from the incomplete detoxification of the cyanide present in tobacco smoke. This failure of detoxification may stem from or be intensified by inadequate dietary intake of necessary nutritional factors. This may be the reason for the association of this disorder with excessive alcohol intake and with its related nutritional deficits (*2, 4, 6, 8, 9, 10, 11, 13, 14, 16, 18, 19, 21, 24, 26, 27, 28*).

## SUMMARY AND CONCLUSIONS

Tobacco amblyopia is presently a rare disorder in the United States. The evidence suggests that this disorder is related to nutritional or idiopathic deficiencies in certain detoxification mechanisms, particularly in handling the cyanide component of tobacco smoke.

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